FOOD MICROSTRUCTURE, Vol. 3 (1984), pp. 127–132
SEM Inc., AMF O'Hare (Chicago), IL 60666-0507 U.S.A.

SOME FACTORS INFLUENCING ANTE-MORTEM CHANGES IN MUSCLE: A BRIEF REVIEW

S.C. Seideman, S.H. Cohen[1] and J.V. Schollmeyer

Roman L. Hruska U.S. Meat Animal Research Center
P.O. Box 166
Clay Center, NE 68933

[1]Science and Advanced Technology Lab
U.S. Army Natick Research and Development Center
Natick, MA 01760

Abstract

Ante-mortem changes to muscle microstructure are influenced in many ways. During pre-natal cell differentiation and growth, myoblasts develop and mature into cells with quite different characteristics. Incorporated into the genome of these cells is the ability to synthesize a wide variety of filaments which occupy specific niches within each myofibril. During post-natal development, depending upon the particular precursor cell lines, different fiber types are produced. These are especially important in contributing to the ultimate palatability of meat. In this paper several factors which influence ante-mortem changes to muscle microstructure are discussed. While some of these are better understood than others, all of them, nevertheless, are important.

Initial paper received January 21 1984.
Final manuscript received November 18 1984.
Direct inquiries to S.H. Cohen.
Telephone number: (617) 651-4578.

Key Words: Growth, muscle cells, intermediate fibers, enzymes, fiber types.

Differentiation and Growth of Muscle Cells

In early stages of prenatal growth, when tissues are differentiating, presumptive myoblasts may undergo a "proliferative" cell cycle, yielding two replicating myoblasts, or may undergo a "quantal" cell cycle which yields one or two postmitotic myoblasts (Holzer et al., 1973). In tissue differentiation, mitosis is the primary mechanism of cell proliferation, and two types of mitosis can occur (see Fig. 1). The first type is proliferative mitosis where presumptive myoblasts replicate. The second type is quantal mitosis where postmitotic myoblasts are formed; i.e., daughter cells may be different from parent cells and cell (tissue) differentiation is initiated.

During early prenatal development, when hyperplasia occurs, mesenchyme cells which are relatively free of thick and thin filaments (Fig. 2), undergo proliferative mitosis. They can also undergo quantal mitosis to form either fibroblasts, the primitive cells of connective tissue, or myoblasts, which are characterized as being mononucleated, but still lacking striations from the precursors of thick and thin filaments (Fig. 3) or, for that matter, striations from the precursors of several classes of intermediate filaments (keratin, desmin, vimentin, etc.) described by Lawson (1983).

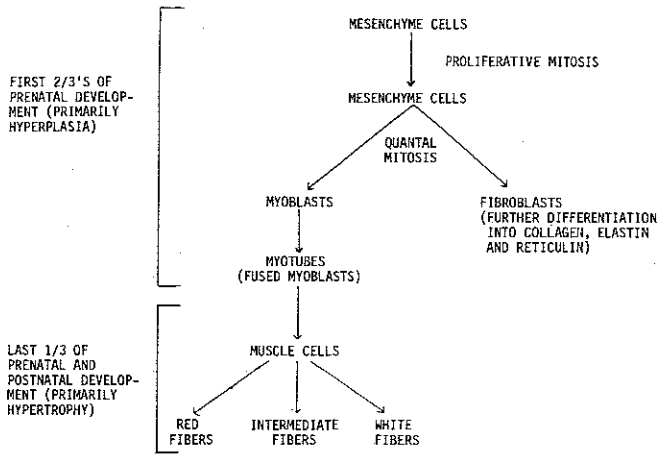
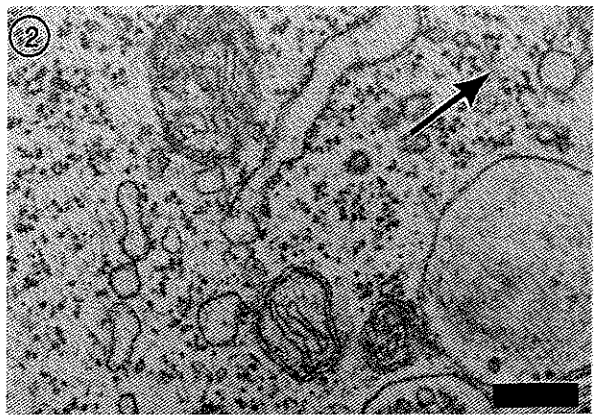
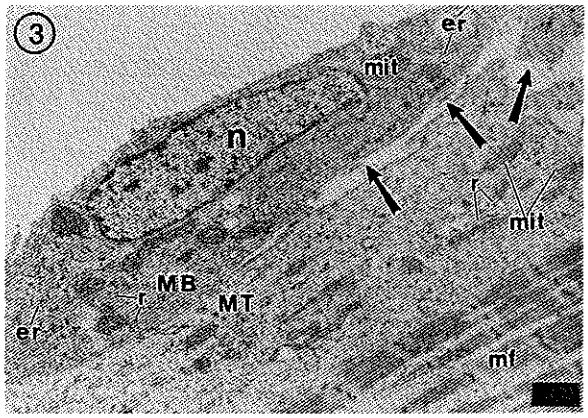
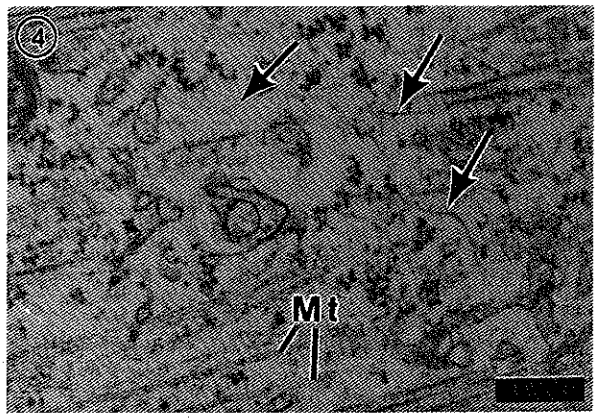


Fig. 1. Differentiation of muscle cells.







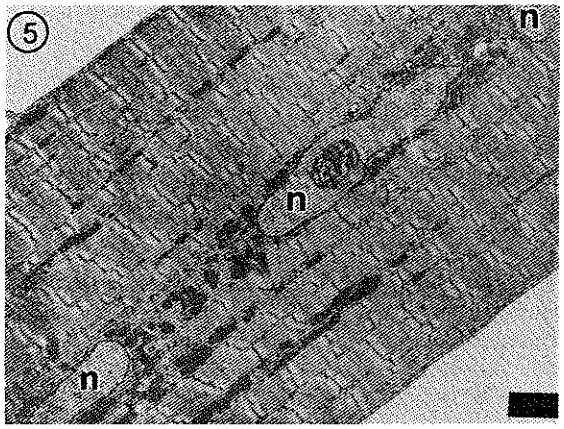


Fig. 2. A mesenchymal cell relatively free of thick and thin filaments has some finer filamentous elements (arrow). Bar = 0.25μm. Reproduced from the Anatomical Record, 1969, vol. 163, page 413. Copyright by the Wistar Institute Press.

Fig. 3. A mononucleated myoblast (MB) is apposed to a myotube (MT) and separated from it on the right side by a space (arrows). Mitochondria (mit), ribosomes and polyribosomes (r), rough-surfaced endoplasmic reticulum (er), a nucleus (n), and myofibrils (mf) can also be seen. Bar = 1 μm. Reproduced from J. Cell Biology, 1971, vol. 48, page 130. By copyright permission of the Rockefeller University Press.

Fig. 4. A myotube (MT) showing some early forming striated myofilaments (arrows). Bar = 0.25 μm. Reproduced from the Cold Spring Harbor Symposia on Quantitative Biology, 1973, vol. XXXVII. Page 554 by copyright permission of the Cold Spring Harbor Laboratory.

Fig. 5. A well developed myotube with nuclei (n). Bar = 0.25 μm. Reproduced from the Cold Spring Harbor Symposia on Quantitative Biology, 1973, vol. XXXVII. Page 552 by copyright permission of the Cold Spring Harbor Laboratory.

Following quantal mitosis, myoblasts interact with one another and fuse to become myotubes (Fig. 4) which ultimately develop into myofibrils and then muscle (Ishikawa et al., 1968). Shortly after undergoing quantal mitosis, the mononucleated myoblasts have synthesized enough myosin, actin and tropomyosin for these proteins to be detected in thick and thin filaments (Holzer et al., 1973).

According to Fischman (1967), the cytoplasmic fusion of mononucleated myoblasts leads to multinucleated skeletal muscle fibers. The fusion is mediated by the Ca^{2+} binding protein, calmodulin, which regulates cyclic nucleotide metabolism, protein phosphorylation, microtube assembly, cell motility, and Ca^{2+} flux across membranes (Bar-Sagi and Prives, 1983). Cells which eventually become myotubes become segregated from surrounding mesenchymal or connective tissue compartments by the acquisition of external lamellae which envelop groups of presumptive muscle cells (Kelly, 1969).

In early myotube development, thick and thin filaments begin to align, then the nuclei from fused myoblasts begin moving to the periphery of the muscle cell (Fig. 5). At this point the entire structure is considered a muscle fiber.

Differentiation of Myofibrillar Filaments

Development of myofibrils precedes formation of the sarcoplasmic reticulum and T-system (Fischman, 1967); however, during myofibrillogenesis two groups of filaments can be found (Kelly, 1968). One is a group of 100 Å diameter filaments with no orientation, the other is a group of 50 Å diameter filaments which aggregate in parallel skeins along the cell membrane. In more mature cells, 130–150 Å filaments, which are probably myosin, can be seen.

Gomer and Lazarides (1983) found that when myoblasts fuse to form myotubes, several proteins including α-actinin, desmin, light and heavy chain myosin, troponin and troponin-T begin to be synthesized. They also found an association of filamin

with the Z line and that the periphery of the Z line also contains actin and intermediate filament proteins such as desmin, vimentin and synemin. α-actinin and another intermediate filament protein—vinculin—are two actin-related proteins which Avnur et al. (1983) found to be located close to the area of microfilament-membrane association.

Several intracellular proteins with apparent organizational roles have been identified and are currently under study. Some of these include (1) fibronectin (Gardner and Fambrough, 1983) which is necessary for the attachment of myoblasts to collagen; (2) C-protein (Offer, 1973) which may be responsible for bridging thick filaments and for myosin regulation; (3) epinemin (Lawson, 1983) which is associated with vimentin filaments in non-neural cells; (4) vinculin (Geiger et al., 1980) which makes up the cortical lattice in skeletal muscle; (5) vimentin (Bennett et al., 1978; Granger and Lazarides, 1978, 1979) which, with desmin, interlinks adjacent myofibrils at the Z line; (6) α-, β- and eu-actinins (Goll et al., 1972; Maruyama, 1976; Kuroda et al., 1981) which are associated with the Z line; (7) M-protein (Masaki et al., 1968) which is a constituent of the M line; (8) synemin (Granger and Lazarides, 1980) which is associated with desmin and vimentin; (9 and 10) titin (Wang et al., 1979) and nebulin (Wang and Williamson, 1980) which are the major components of the longitudinal filaments which connect the Z lines.

The study of the antemortem synthesis of these proteins is essential for a thorough understanding of the conversion of muscle to meat.

Postnatal Development of Muscle Cells

Hypertrophy characterizes postnatal development, with muscle fibers growing by increasing in both diameter and length. The rate of size increase slows as an animal approaches maturity. Forrest et al. (1975) found the diameter of individual muscle fibers is increased as myofibrils proliferate by longitudinal splitting of larger myofibrils into smaller ones. This causes the number of myofibrils within a fiber to increase by 10–15 times during an animal's lifetime.

The length of a muscle is increased by two mechanisms; firstly, by the fusion of myogenic cells with the existing muscle fiber; and secondly, by the generation of new contractile units, the sarcomeres, which are inserted into myofibrils where the fiber ends attach to connective tissue (Moss and LeBlond, 1971).

Muscle Fiber Types

There is evidence that differentiation into diverse fiber types occurs after the formation of muscle fibers, not before (Stockdale, 1982). Some unknown process, either endogenous or exogenous, causes the embryonic muscle fiber to change its genomic programming which leads to the differentiation of individual fiber types (Toyota and Shimada, 1981).

Red fibers are usually the ones present at birth and transform into intermediate and white fibers (Rowe and Goldspink, 1969). Tomanek (1976) reported that, in postnatal development, red fibers differentiate first into white and finally into intermediate fibers. There is a gradual transition from red to white fibers as an animal matures (Dreyer et al., 1977).

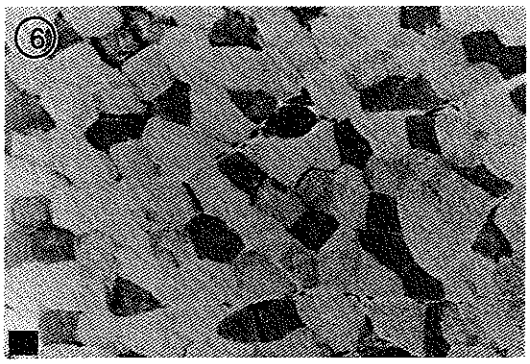
Physiologically, according to Squire (1981), mammalian muscle fibers can be distinguished by whether their energy supply is glycolytic or oxidative. Glycolytic fibers are fast contracting and appear white; oxidative fibers have a slow contractile response and appear red. These slow and fast fibers are referred to as Type I and II respectively and the Type II fast fibers are subdivided into fast oxidative/glycolytic (IIA) and fast glycolytic (IIB).

Table 1. Characteristics of Red and White Muscle Fibers

Red Fibers
Contains more myoglobin
Sudan black B positive (more lipids)
More mitochondria
Smaller in diameter
In clumps that are surrounded by white fibers (pig)
Surrounded by many capillaries
Rich in sarcoplasm
Less soluble protein
Less connective tissue
Aerobic metabolism
High oxidative enzyme activity (TCA cycle activity)
Contains less glycogen
Slow contraction
Muscles: Psoas
Soleus
Trapezius
Sartorius
White Fibers
Contains less myoglobin
Sudan black B negative (less lipids)
Fewer mitochondria
Larger in diameter
In periphery of bundle (pig)
Surrounded by fewer capillaries
Less sarcoplasm
More soluble protein
More connective tissue
Anaerobic metabolism
High glycolytic enzyme activity (glycogen and glucose activity)
Contains more glycogen
Twitch contraction (fast)
Muscles: Longissimus
Brachioradialis
Gastrocnemius

Ultrastructurally, there are three types of fibers which are recognized in fast twitch muscle. They are: white fibers which have narrow Z lines and few mitochondria; intermediate fibers which have narrow Z lines and many mitochondria; and red fibers which have both wide Z lines and many mitochondria (Gauthier, 1979). Some of the characteristics of red and white fibers are summarized in Table 1.

Fig. 6 shows the three fiber types based on their NADH reductase content. The red fibers stain dark, white fibers stain white and intermediate fibers stain less intensely than the red fibers. In addition to differing enzymatically, the ultrastructural differences are seen in Figs. 7, 8 and 9, which are electron micrographs of red, intermediate and white fibers, respectively. The graded concentration of mitochondria and Z line differences can also be distinguished.



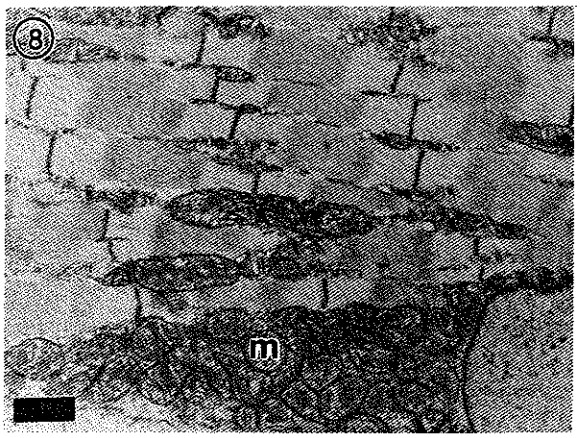
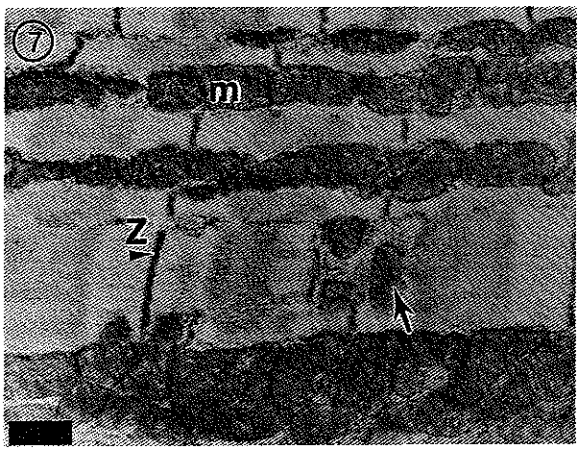


Fig. 6. Light micrograph of three fiber types (strong, weak and intermediate staining) based on $NADH_2$-tetrazolium reductase. Bar = 50 μm. Reproduced from Dubovitz, V. 1970. Differentiation of fiber types in skeletal muscle. In "The Physiology and Biochemistry of Muscle as a Food, 2." Ed. by EJ Briskey, RG Cassens, BB Marsh. Page 90 by copyright permission from the University of Wisconsin Press.

Fig. 7. Electron micrograph of a red muscle fiber. The Z line (Z), large mitochondria with closely packed cristae (m) and a portion of paired mitochondria (arrow) can be seen. Bar = 1 μm. Reproduced from Gauthier, GF, 1970. The ultrastructure of three fiber types in mammalian muscle. In "The Physiology and Biochemistry of Muscle as a Food, 2." Ed. by EJ Briskey, RG Cassens, BB Marsh. Page 110 by copyright permission from the University of Wisconsin Press.



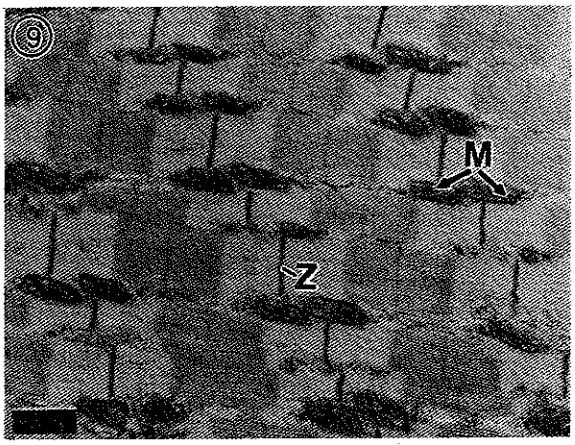


Fig. 8. Electron micrograph of an intermediate muscle fiber. The intermediate fiber is similar to the red fiber in Fig. 7 except the mitochondria (m) are somewhat smaller and the Z lines are narrower. Bar = 1 μm. Reproduced from Gauthier, GF. 1970. In "The Physiology and Biochemistry of Muscle as a Food, 2." Ed. by EJ Briskey, RG Cassens, BB Marsh. Page 111 by copyright permission from the University of Wisconsin Press.

Fig. 9. Electron micrograph of a white fiber. Note the paired mitochondria (M) and relatively thin Z lines (Z). Bar = 1 μm. Reproduced from Gauthier, GF. 1970. In "The Physiology and Biochemistry of Muscle as a Food, 2." Page 112 by copyright permission from the University of Wisconsin Press.

Factors Influencing Muscle Mass

Since the number of fibers is the most important factor in limiting ultimate muscle size (Goldspink, 1974), and since the genetically determined number is achieved at or near the time of birth, increase in muscle mass is accomplished only by enlargement of muscle fibers (Luft and Goldspink, 1967). All muscle fibers enlarge, but particularly by the conversion of red fibers to larger white fibers.

Spindler et al. (1980) reported that as cattle aged, mean cross sectional area of fibers doubled overall, while the cross sectional area of white fibers increased slightly, and red fibers decreased slightly. Ashmore et al. (1972) evaluated growth patterns in piglet, lamb and calf muscles histochemically and concluded that transformation of intermediate fibers to white fibers increased at the expense of red fibers without alteration in the total number of fibers.

Reducing food intake leads to a reduction in muscle mass, since starvation causes a decrease in fiber size and, at the same time leads to a reduction in the number of myofibrils (Van Linge, 1962). Decrease in muscle bulk is directly related to a decrease in mean fiber diameter in cattle (Robertson and Baker, 1933) and in pigs (McMeekan, 1940).

Biochemical control of protein metabolism in relation to starvation is not completely understood; however, it appears that

not only is the rate of protein degradation increased during starvation, but the rate of synthesis of new protein is decreased as well (Young, 1970). This may result from a shortage of one or more essential amino acids in the muscle fibers or it may be due to a reduction in the protein synthesizing capacity of polyribosomes.

Moody et al. (1980) postulated that the nature of the available source of energy in lamb rations caused a shift from intermediate to white muscle fibers. As the protein content of rations increased, percentages of intermediate and white fibers decreased quadratically (Johnston et al., 1975).

In general, there is no difference in the total number of muscle fibers in the same anatomical muscles of males and females; however, even though fiber size is greater in males, Brooke (1970) reported fiber type percentage difference is similar for both sexes. But, he found white fibers are smaller than red fibers in females and larger in males. On the other hand, Dreyer et al. (1977) reported that bulls had a higher percentage of red fibers and a lower percentage of white fibers than steers. This contrasts with results obtained by Bass et al. (1971) who found that significant growth occurred in guinea pig temporal muscle following administration of testosterone, and, this growth was accompanied by a change from intermediate to white fibers.

Miscellaneous Factors

At the present time, selection of meat animals is based upon the appraisal of such characteristics as muscle size and growth rate (Swatland, 1973). Being blind to all the factors contributing to muscle size and growth rate, it is not surprising that intensive selective breeding can produce or reveal anomalies such as stress susceptible pigs or double muscled cattle. Stress susceptible pigs have a higher white to red fiber ratio compared to normal pigs (Didley et al., 1970) and double muscled cattle have larger and more abundant white fibers than red, compared with normal cattle (Holmes and Ashmore, 1972).

Several researchers have related fiber diameter and muscle bundle size to meat quantity. Joubert (1956) and Hegarty (1971) reported that fiber diameter increases with age in cattle. Miller et al. (1975) found the total number of muscle fibers was more closely related to muscle mass than was fiber diameter. In addition, the authors found that faster growing pigs appeared to possess more, but smaller muscle fibers than slower growing pigs. Castle and Gregory (1929), Smith (1963) and Staun (1963) found just the opposite to be the case. They reported that animals possessing large muscle fibers are often rapidly growing and muscular.

Calkins et al. (1981) related palatability to fiber type and reported that the presence of white fibers correlated poorly with marbling and tenderness ratings; however, the presence of red fibers correlated well. The authors concluded that, since the oxidative capacity of a muscle is related to marbling and tenderness, fiber type could possibly be used to predict both marbling and tenderness.

Conclusion

Ante-mortem changes in muscle obviously affect the ultimate palatability of meat; however, it is not just one event occurring prior to slaughter which determines how tough or tender meat will be. Many factors have to be considered as being potentially responsible for determining ultimate acceptability. The variations in a given muscle between individual animals, sexes and species exacerbate the problem. There are just so many factors, a solution awaits further research.

References

Ashmore CR, Tompkins G, Doerr L. (1972). Postnatal development of muscle fiber types in domestic animals. J Anim. Sci. **34**:37–41.

Avnur Z, Small JV, Geiger B. (1983). Actin-independent association of vinculin with the cytoplasmic aspect of the plasma membrane in the cell-contact areas. J. Cell Biol. **96**:1622–1630.

Bar-Sagi D, Prives J. (1983). Trifluoperazine, a calmodulin antagonist inhibits muscle cell fusion. J. Cell Biol. **97**:1375–1380.

Bass A, Gutmann E, Hanzlikova V, Syrovy L. (1971). Sexual differentiation of enzyme pattern and its conversion by testosterone in the temporal muscle of the guinea pig. Physiol. Biochem. **20**:423–431.

Bennett GS, Fellini SA, Croop JM, Otto JJ, Bryant J, Holzer H. (1978). Differences among the 100 A-filament subunits from different cell types. Proc. Natl. Acad. Sci. **75**:4364–4368.

Brooke MH. (1970). Some comments on neural influence on the two histochemical types of muscle fibers. In: "Physiology and Biochemistry of Muscle as a Food" Ed. by EJ Briskey, RG Cassens, BB Marsh. Univ. of Wisconsin Press, Madison, WI, pp. 131–153.

Calkins CR, Dutson TR, Smith GC, Carpenter ZL, Davis GW. (1981). Relationship of fiber type composition to marbling and tenderness of bovine muscle. J. Food Sci. **46**:708–710.

Castle WE, Gregory PW. (1929). The embryological basis of size inheritance in the rabbit. J. Morphol. and Physiol. **48**:81–103.

Didley DD, Aberle ED, Forrest JC, Judge MD. (1970). Porcine muscularity and properties associated with pale, soft, exudative muscle. J. Anim. Sci. **31**:681–685.

Dreyer JH, Naude RT, Henning JWN, Rossouw E. (1977). The influence of breed, castration and age on muscle fiber type and diameter in Friesland and Afrikaner cattle. S. Afr. J. Anim. Sci. 7:171–180.

Fischman DA. (1967). An electron microscope study of myofibril formation in embryonic chick skeletal muscle. J. Cell Biol. **32**:557–575.

Forrest JC, Aberle ED, Hedrick HB, Judge MD, Merkel RA. (1975). Principles of Meat Science. WH Freeman and Co., San Francisco, CA, pp. 82–90.

Gardner JM, Fambrough DM. (1983). Fibronectin expression during myogenesis. J. Cell Biol. **96**:474–485.

Gauthier GF. (1979). Ultrastructural identification of muscle fiber types by immunocytochemistry. J. Cell Biol. **82**:474–485.

Geiger B, Tokuyasu KT, Dutton AH, Singer SJ. (1980). Vinculin, an intracellular protein localized at specialized sites where microfilament bundles terminate at cell membranes. Proc. Natl. Acad. Sci. **77**:4127–4131.

Goldspink G. (1974). Development of muscle. In: "Differentiation and Growth of Cells in Vertebrate Tissue" Ed. by G. Goldspink. Halsted Press (Div. of John Wiley and Sons), New York, pp. 69–99.

Goll DE, Suzuki A, Temple J, Holmes GR. (1972). Studies on purified α-actinin/F-actin interaction. J. Mol. Biol. **67**:469–488.

Gomer RH, Lazarides E. (1983). Switching of filamin polypeptide during myogenesis in vitro. J. Cell Biol. **96**:321–329.

Granger BL, Lazarides E. (1978). The existence of an insoluble Z disc scaffold in chicken skeletal muscle. Cell. **15**:1253–1268.

Granger BL, Lazarides E. (1979). Desmin and vimentin coexist at the periphery of the myofibril Z disc. Cell. **18**:1053–1063.

Granger BL, Lazarides E. (1980). Synemin: Two new high molecular weight proteins associated with desmin and vimentin filaments in muscle. Cell. **22**:727–738.

Hegarty PVJ. (1971). Muscle fiber growth and development. Proc. Recip. Meat Conf., National Livestock and Meat Board, Chicago, IL, **24**:319–349.

Holmes JHG, Ashmore CR. (1972). A histochemical study of development of muscle fiber type and size in normal and double muscled cattle. Growth. **36**:351–372.

Holzer H, Sanger JW, Ishikawa H, Strahs K. (1973). Selected topics in skeletal muscle myogenesis. Cold Spring Harbor Symp. on Quant. Biol. **37**:549–566.

Ishikawa H, Bischoff R, Holzer H. (1968). Mitosis and intermediate-sized filaments in developing skeletal muscle. J. Cell. Biol. **38**:538–555.

Johnston DM, Stewart DF, Moody WG, Boling JA, Kemp JD. (1975). Effect of breed and time on the size and distribution of beef muscle fiber types. J. Anim. Sci. **40**:613–620.

Joubert DM. (1956). An analysis of factors influencing postnatal growth and development of the muscle fiber. J. Agric. Sci. **47**:59–102.

Kelly DE. (1968). Myofibrillogenesis and the differentiation of Z-bands in developing amphibian skeletal muscle. Anat. Rec. **160**:474.

Kelly DE. (1969). Myofibrillogenesis and Z-band differentiation. Anat. Rec. **163**:403–426.

Kuroda M, Tanaka T, Masaki T. (1981). Eu-actinin, a new structural protein of the Z-line of striated muscle. J. Biochem. **89**:297–310.

Lawson D. (1983). Epinemin: a new protein associated with vimentin filaments in non-neural cells. J. Cell Biol. **97**:1891–1905.

Luft AR, Goldspink G. (1967). Large and small muscles. Life Sci. **6**:1821–1826.

Maruyama K. (1976). Actinins, regulatory proteins of muscle. Adv. Biophys. **9**:157–185.

Masaki T, Takaiti S, Ebashi S. (1968). "M-substance", a new protein constituting the M-line of myofibrils. J. Biochem. **71**: 355–357.

McMeekan CP. (1940). Growth and development of the pig with special reference to carcass quality characteristics. J. Agri. Sci. **30**:276–344.

Miller LR, Garwood VA, Judge MD. (1975). Factors affecting porcine muscle fiber type, diameter and number. J. Anim. Sci. **41**:66–77.

Moody WG, Kemp JD, Mahyuddin M, Johnston DM, Ely DG. (1980). Effect of feeding systems, slaughter weight and sex on histological properties of lamb carcasses. J. Anim. Sci. **50**: 249–256.

Moss FP, LeBlond CP. (1971). Satellite cells as a source of nuclei in muscles of growing rats. Anat. Rec. **170**:421–435.

Offer G. (1973). C-protein and the periodicity in the thick filaments of vertebrate skeletal muscle. Cold Spring Harbor Symp. Quant. Biol. **37**:87–93.

Robertson JD, Baker DD. (1933). Histological differences in the muscles of full and rough fed steers. Univ. of Michigan Res. Bull. #200, pp. 1–11, Ann Arbor, MI.

Rowe RWD, Goldspink G. (1969). Muscle fiber growth in five different muscles in both sexes of mice. I. Normal mice. J. Anat. **104**:519–530.

Smith JH. (1963). Relation of body size to muscle cell size and number in the chicken. Poul. Sci. **42**:283–293.

Spindler AA, Mathias MM, Cramer DA. (1980). Growth changes in bovine muscle fiber types as influenced by breed and sex. J. Food Sci. **45**:29–31.

Squire J. (1981). The Structural Basis of Muscular Contraction. Plenum Press, New York, 698 pp.

Staun H. (1963). Various factors affecting number and size of muscle fibers in the pig. Acta Agric. Scand. XIII, 293–322.

Stockdale FE. (1982). Introduction: Myoblast commitment and the embryogenesis of skeletal muscle. In: "Muscle Development, Molecular and Cellular Control." Ed. by ML Pearson, HF Epstein. Cold Spring Harbor Laboratory, NY, pp. 339–344.

Swatland HJ. (1973). Muscle growth in the fetal and neonatal pig. J. Anim. Sci. **37**:536–545.

Tomanek RJ. (1976). Ultrastructural differentiation of skeletal muscle fibers and their diversity. J. Ultrastructure Res. **55**: 212–227.

Toyota N, Shimada Y. (1981). Differentiation of troponin in cardiac and skeletal muscles in chicken embryos as studied by immunoflorescence microscopy. J. Cell Biol. **91**:497–504.

Van Linge B. (1962). The response of muscle to strenuous exercise. J. Bone and Joint Surg. **44**:711–721.

Wang K, McClure J, Tu A. (1979). Titin: Major myofibrillar components of striated muscle. Proc. Natl. Acad. Sci. **76**: 3698–3702.

Wang K, Williamson CL. (1980). Identification of an N_2 line protein of striated muscle. Proc. Natl. Acad. Sci. **77**:3254–3258.

Young VR. (1970). The role of skeletal and cardiac muscle in the regulation of protein metabolism. In: "Mammalian Protein Metabolism." Ed. by Munro HN, Academic Press, New York, **4**:586–674.

Discussion with Reviewers

Reviewer III: What is the definition of mitosis in the context of this review?

Authors: Typically, mitosis is the resolution of nuclear chromatin into a thread-like form that separates into chromosomes. Each chromosome divides, resulting in two new cells. What distinguishes myoblasts from other cells which undergo mitosis is the type of contractile protein precursor present. These embryonic proteins may be preferentially distributed among different cells during the cell cycle.

Reviewer II: Please elaborate on features shown in Fig. 2.

Authors: At the stage of the cell cycle shown in this figure the filaments have just begun to be synthesized. Since only a very few filaments can be seen, we assume that the photomicrograph is of a relatively immature cell.

Reviewer I: Could you please explain what is the difference in taste between the meat of a normal pig and that of a stress susceptible pig?

Authors: The meat which comes from a stress susceptible pig is pale, soft and exudative or PSE. When this type of meat is cooked, because it has a decreased water holding capacity, it is much less juicy than normal.